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Mortality burdens in California due to air pollution attributable to local and nonlocal emissions

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Abstract

Limited research has been conducted on the contributions of local and nonlocal emission sources to ambient fine particulate matter ($PM_{2.5}$) and ozone (O_3) and their associated mortality. In this study, we estimated the total mortality resulting from long-term $PM_{2.5}$ and O_3 exposures in California in 2012 using multiple concentration response functions (CRFs) and attributed the estimated mortality to different emission groups. The point estimates of $PM_{2.5}$-associated mortality in California ranged from 12,700 to 26,700, of which 53% were attributable to in-state anthropogenic emissions. Based on new epidemiological evidence, we estimated that O_3 could be associated with up to 13,700 deaths from diseases of both the respiratory and cardiovascular systems in California. In addition, 75% of the ambient O_3 in California was due to distant emissions outside the western United States, leading to 92% of the O_3-associated mortality. Overall, distant emissions lead to greater mortality burdens of air pollution in California than local anthropogenic emissions.

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Appendix A. Supplementary material
Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.105232.

Keywords

Emissions; Fine particulate matter; Ozone; Mortality

1. Introduction

Air pollution is known to have multiple adverse effects on human health (Pope and Dockery, 2006; Hoek et al., 2013; Kim et al., 2015). In particular, mortality due to exposure to fine particulate matter ($PM_{2.5,}$ particles with aerodynamic diameters of 2.5 μm or less) and tropospheric ozone (O_3) remains a major global concern. Of the two pollutants, $PM_{2.5}$ was found to dominate the mortality burden and health cost estimates, especially at the national and global levels (Anenberg et al., 2010; Cohen et al., 2017; Fann et al., 2012; U.S. EPA, 2012). Over the past decades, extensive efforts have been made to understand the effects of long-term exposure to $PM_{2.5}$ (Krewski et al., 2009; Hoek et al., 2013; Turner et al., 2016; Burnett et al., 2014, 2018) and O_3 (Jerrett et al., 2009; Turner et al., 2016) on mortality. With increased cohort data and refined epidemiological models, newer studies have shown different concentration-response functions (CRFs) from previous studies with respect to individual air pollutants. The impacts of updated CRFs on mortality estimates, however, have rarely been quantified. With respect to O_3, earlier studies restricted burden estimates to respiratory mortality, but recent studies have reported that O_3 also contributes to all-cause and cardiovascular mortality (Crouse et al., 2015; Turner et al., 2016). Toxicological evidence also suggests that O_3 exposure can elicit cardiovascular health effects (Devlin et al., 2012).

California is the most populous state in the United States (i.e., 39.5 million in 2017) and the world's fifth-largest economy in 2018 (California Department of Finance, 2019). Although California has aggressively controlled air pollution over the past 50 years, it is still the home to seven of the top ten U.S. cities with the most severe $PM_{2.5}$ pollution and eight of the ten worst U.S. cities for O_3 pollution (Billings et al., 2018). Because of the population and economy, emissions generated from local human activities contributed greatly to the air pollution in California, but distant emissions from other states or countries can also significantly affect the ambient $PM_{2.5}$ and O_3 concentrations in this coastal state along the Pacific Ocean (Chin et al., 2007; Heald et al., 2006; Yu et al., 2012). Background ozone, which is defined as the O_3 concentrations that would occur in the absence of local anthropogenic emissions, has been found to contribute greatly to ambient O_3 in California (Emery et al., 2012; Fiore et al., 2014; Huang et al., 2015). As California continues to clamp down on local emissions (California Air Resources Board, 2017), the impacts of nonlocal emissions through long-range transport could become more significant in the future. Previous studies, however, have not systematically analyzed the contributions of local and nonlocal emissions to ambient $PM_{2.5}$ and O_3 concentrations in California or their associated health impacts.

In this study, we aim to (1) assess the relative impacts of local and nonlocal emissions on air quality and mortality in California and (2) compare air pollution-associated disease burdens

from multiple epidemiological models, including new models that suggest greater risks for mortality in association with long-term exposure to $PM_{2.5}$ and O_3.

2. Materials and methods

2.1. Study design

In this study, we first designated all emissions affecting ambient $PM_{2.5}$ and O_3 in California into four emission groups based on geographic regions (illustrated in Fig. 1): (1) California in-state anthropogenic emissions, including power plants, industry, residential and commercial, transportation, agriculture, solvent use, and crop residue burning; (2) anthropogenic emissions from other western United States, excluding California; (3) natural emissions from the western United States (including California), including wind-blown dust, biogenic nonmethane volatile organic compounds (NMVOCs), sea salt, wildfire, and lightning NOx emissions; and (4) all emissions from outside of the western United States. Second, by using the Weather Research and Forecasting Model with Chemistry (WRF-Chem), we modeled seasonal hypothetical ambient $PM_{2.5}$ and O_3 concentrations if any one of the four emission groups are turned off (i.e., four season × four emission groups). The difference between baseline and each hypothetical scenario therefore represents the air pollution contributions of the respective emission group that was turned off. Finally, we estimated overall $PM_{2.5}$ and O_3-associated mortality burdens, as well as the mortalities that are attributed to individual emission groups. Mortality burdens of air pollution were monetized following the health impact analyses to estimate the monetary value of health-associated public losses.

2.2. Air quality modeling

We simulated the $PM_{2.5}$ and O_3 concentrations using the WRF-Chem model (version 3.9.1) over a domain covering the western U.S. and its surrounding areas at a 12 km × 12 km horizontal resolution. The vertical resolution of WRF-Chem includes 24 layers from the surface to 100 hPa, with denser layers at lower altitudes to resolve the planetary boundary layer (PBL). The meteorological initial and boundary conditions were generated from the Final Operational Global Analysis data (ds083.2) of the National Center for Environmental Prediction (NCEP) at a 1.0° × 1.0° and 6-h resolution. The chemical initial and boundary conditions were extracted from the output of the Model for Ozone and Related Chemical Tracers version 4 (MOZART-4) (Emmons et al., 2010). A 6-day spin-up period was used to minimize the influence of the initial conditions on the simulation results. We employed an extended Carbon Bond 2005 (CB05) (Yarwood et al., 2005) with chlorine chemistry (Sarwar et al., 2008) coupled with the Modal for Aerosol Dynamics in Europe/Volatility Basis Set (MADE/VBS) (Ahmadov et al., 2012; Wang et al., 2015). MADE/VBS uses a modal aerosol size representation and an advanced secondary organic aerosol (SOA) module based on the VBS approach. This model also considered aerosol direct radiative effects and first and second aerosol indirect effects on gridscale clouds following our previous study (Zhao et al., 2017a). Other physical and chemical options used in the model are described in the Supplementary Information.

Considering a large computational burden, the simulation periods were held in January, April, July, and October 2012, representing seasonal air pollution levels in winter, spring, summer, and fall, following previous studies (Wang et al., 2015; Zhao et al., 2017b). The year 2012 was selected for modeling in consideration of data consistency with California's state emission inventory (i.e., the California Emission Projections and Analysis Model, or CEPAM). Although the exact emissions of individual sources may have changed slightly since 2012, the analysis presented in this paper would still be informative for future decision making and planning practices because the general source contribution pattern is likely to continue in the near future. For anthropogenic emissions, we used the National Emission Inventory (NEI). The NEI is updated only approximately every three years, and 2011 is the closest year to our simulation period. The air pollutant emissions changed slightly from 2011 to 2012, and we scaled the NEI 2011 inventory to the 2012 levels according to the *NEI trend report* (U.S. EPA, 2016). The data sources of biogenic emissions, dust emissions, seasalt emissions, and plume rise of wildfire are provided in the Supplementary Information.

We evaluated simulated daily average concentrations of $PM_{2.5}$ and their major chemical composition and daily maximum 1-h and 8-h O_3 concentrations against surface observations from three monitoring networks: the Air Quality System (AQS), the Interagency Monitoring of Protected Visual Environments (IMPROVE), and the Clean Air Status and Trends Network (CASTNET) (see Supplementary Information Fig. S1 and Table S2). The meteorological predictions were compared with observational data obtained from the National Climatic Data Center (NCDC) (see Supplementary Information Table S1).

2.3. Spatial source contribution analysis

We first developed a baseline scenario to represent the real-world emissions and air quality conditions in four seasons. Four hypothetical emission scenarios were also designed in WRF-Chem based on the four emission groups shown in Fig. 1: *Scenario 1*, which turns off all California in-state anthropogenic emissions, represents the impact of local emissions (i.e., to model air quality if there were no local emissions).*Scenario 2*, which turns off anthropogenic emissions from other western U.S. states, represents the impact of regional emissions. *Scenario 3*, which turns off natural emissions in the western United States (including California), represents the natural impact in the region. *Scenario 4*, which turns off all emissions from Canada, Mexico, and U.S. states other than the western United States within the modeling domain and sets the chemical boundary condition to zero, represents the impact of global emissions. The modeled ambient $PM_{2.5}$ (and O_3) concentration differences between each of the four hypothetical scenarios and the baseline scenario represent the contribution of the corresponding emission group to ambient air quality. The relative contribution (unit: %) of an emission group was calculated using the arithmetic mean of the concentration contribution (unit: $\mu g/m^3$ for $PM_{2.5}$, ppb for O_3) in all California grid cells divided by the baseline state average air pollution concentration. Because of the complex nonlinear emission-concentration relationships, the percent contributions of four emission groups does not necessarily add up to 100%. The simple add-up of contributions from all emission groups was higher than the baseline concentration for $PM_{2.5}$ and lower for O_3. The method used in this study to quantify source contributions has been the most widely used method in previous studies (Zhao et al., 2015).

2.4. Health impact analysis

Mortalities were estimated based on ambient air pollutant concentrations using concentration response functions (CRFs) derived from previous epidemiological studies. Four $PM_{2.5}$ CRFs were analyzed: two log-linear functions for all-cause mortality derived from Krewski et al., (2009) and Hoek et al., (2013), respectively, the integrated exposureresponse (IER) model (Burnett et al., 2014), and the recently developed Global Exposure Mortality Model (GEMM) (Burnett et al., 2018). Three O_3 CRFs were analyzed: one for all-cause mortality (Turner et al., 2016) and two for respiratory mortality (Jerrett et al., 2009; Turner et al., 2016). The detailed CRF parameters used in our study are listed in the Supplementary Information (Tables S3). Health impact analyses were conducted using the Environmental Benefit Mapping and Analysis Program (BenMAP-CE, version 1.3.7) developed by the U.S. EPA. Gridlevel demographic data and baseline all-cause mortality rates were obtained from the default database in BenMAP. Baseline mortality rates in California for noncommunicable diseases (NCDs), lower respiratory infections (LRIs), and cardiovascular diseases (CVDs) were obtained from the GBD database (http://ghdx.healthdata.org/gbd-results-tool, see Supplementary Information). The total $PM_{2.5}$- and O_3-associated mortality burdens in California under log-linear CRFs were the aggregate amount of mortality impacts of individual emission groups. The total mortality burden under IER and GEMM was directly estimated using the baseline scenario due to the nonlinear shape of the mortality CRFs. The 95% confidence intervals (CIs) were calculated using Monte-Carlo analysis based on the uncertainty in the parameters of CRFs. We next combined individual $PM_{2.5}$ and O_3 CRFs to generate 12 combinations and calculated the total air pollution-related mortality burdens and the relative contribution of nonlocal emissions for each combination. Nonlocal emissions include the other three emissions groups except for the in-state anthropogenic emissions in the study.

We applied the “value of statistical life (VSL)” approach (U.S. EPA, 2018) to evaluate the change in premature mortality risk due to air pollution exposure. The unit value of the VSL was assumed to be 8.4 million U.S. dollars (USD, with the 2012 inflation rate). This is an intermediate value of many studies and is consistent with the U.S. EPA’s Regulatory Impact Analyses (RIAs) and the Section 812 Retrospective and Prospective Analyses of the Clean Air Act (U.S. EPA, 2018).

3. Results

We found that in-state anthropogenic emissions and distant emissions from outside of the western United States both significantly contributed to ambient $PM_{2.5}$ in California with moderate seasonal variations (Fig. 2a). In-state anthropogenic emissions contributed 47% (2.3 $\mu g/m^3$) to the annual average ambient $PM_{2.5}$ concentration in California, and distant sources contributed to 48% (2.4 $\mu g/m^3$), followed by natural emissions from the western United States (i.e., 1.1 $\mu g/m^3$) and anthropogenic emissions in the western United States, excluding California (i.e., 0.3 $\mu g/m^3$). In contrast, ambient O_3 concentrations in California were predominantly determined by distant emissions from outside of the western United States in all seasons, with an annual average of 29.8 ppb (75%) (Fig. 2b). Its relative contributions range from 53% in summer to 97% in winter (Fig. 2b). In-state emissions have

highly heterogeneous seasonal impacts on ambient O_3. They contributed the most in summer (23% or 10.7 ppb), while in winter, their contribution was negative (i.e., −2%), possibly due to the NMVOC-limited photochemical regime. The contribution of individual emission groups includes both primary and secondary impacts and is thus nonlinear with respect to baseline total $PM_{2.5}$ and O_3.

We then analyzed the $PM_{2.5}$- and O_3-associated mortality burdens in California disaggregated by different emission groups, as summarized in Fig. 3. The highest estimate showed that $PM_{2.5}$ was associated with 26,700 (95% CI: 18,800–35,000) deaths in California in 2012, which is based on the CRF derived from the recent GEMM (Burnett et al., 2018), while the lowest estimate was 12,700 (95% CI: 3100– 24,300) using the IER (Fig. 3a). For O_3, by using the CRFs derived from Turner et al., (2016), we estimated that the long-term exposure was responsible for 13,700 (95% CI: 6100–23,700) deaths in California in 2012, of which 7300 (95% CI: 2900–11,000) were diseases of the respiratory system (Fig. 3b). In addition to respiratory mortality, this model also estimated that long-term O_3 exposure was associated with 6400 (95% CI 2200–10,300) deaths from CVDs (Table S4). The CRF derived from Jerrett et al., (2009), which was between respiratory mortality and the 1-h maximum O_3 concentration, estimated much smaller mortality burdens of O_3 [i.e., 3300 (95% CI: 1100–5300)]. We then compared the mortality estimates under different combinations of $PM_{2.5}$ and O_3 CRFs (Fig. 3c). Overall, point estimates of air pollution-associated mortality for different CRF combinations varied between 16,000 and 40,400 deaths per year. Fig. 3c also analyzed the relative impacts of local vs. nonlocal emissions on total mortality burdens and found that the relative contribution of nonlocal emissions (i.e., all emissions except for California in-state anthropogenic emissions) ranged from 48% to 70% under different $PM_{2.5}$ and O_3 CRF combinations.

The “worst-case scenario”, which accounted for the estimated $PM_{2.5}$-associated mortality from the GEMM and O_3-associated mortality of all causes from the Turner CRF, was then further analyzed. Under this scenario, ambient $PM_{2.5}$ originating from California in-state anthropogenic emissions has led to the most deaths [i.e., 14,000 (95% CI 9,900–18, 400)], accounting for 53% of the mortality burden attributable to $PM_{2.5}$. Meanwhile, we found that distant emissions out of the western U.S. were responsible for 92% of O_3-associated mortality. The monetized health losses due to $PM_{2.5}$ and O_3 exposures are estimated at $224 billion and $115 billion, respectively. The breakdown of monetized mortality costs by emission groups is listed in the Supplementary Information (Table S5).

We next analyzed the spatial distributions of ambient air pollution concentration contributions and the associated mortality burdens under the worst-case scenario for key emission groups. Ambient $PM_{2.5}$ originating from in-state anthropogenic emissions (Fig. 4a) shows a distinct spatial pattern from distant emissions out of the western United Statesinduced $PM_{2.5}$ (Fig. 4c). Although the two emission groups contributed almost equally to the statewide average $PM_{2.5}$ concentrations, in-state anthropogenic emissions-induced $PM_{2.5}$ was mainly concentrated in urban areas with dense populations. Consequently, $PM_{2.5}$ originating from in-state anthropogenic emissions led to much more deaths in Los Angeles, the San Francisco Bay Area, and the San Juaquin Valley (Fig. 4b and d). In terms of O_3, our model demonstrates that California in-state emissions negatively affect ambient O_3 (Fig. 5a)

and the associated deaths (Fig. 5b) in Los Angeles and San Francisco. O_3 originating from distant emissions out of the western United States was rather uniformly distributed across the state (Fig. 5c) and led to the most O_3-associated deaths (Fig. 5d). The spatial distributions of mortality burdens illustrated in Figs. 4 and 5 both indicated that air pollution-associated mortalities in California were mainly concentrated in metropolitan areas, namely, the Los Angeles Basin and the San Francisco Bay Area.

4. Discussion

Air pollution is known to adversely affect human health through multiple pathways. Previous studies suggested that chronic exposure to $PM_{2.5}$ accounted for the vast majority of excess deaths (Anenberg et al., 2010; Cohen et al., 2017; Fann et al., 2012; Xie et al., 2019). Therefore, an important implication of our study is that O_3-associated mortality might be underestimated in past studies. We find that the O_3-associated mortality burden is comparable to the $PM_{2.5}$-associated mortality burden in California based on new epidemiological evidence. By conducting a spatial source contribution analysis, we also find that overall, distant emissions contribute more to ambient O_3 concentrations and air pollution-related deaths in California than local anthropogenic emissions (Fig. 3).

The mortality burden estimates in California are higher using newer epidemiological models than previous ones, suggesting that improving air quality in California could potentially lead to more health benefits than previously thought. Meanwhile, the large variations in mortality estimates under different CRFs also indicate that the uncertainties inherent to the existing health impact analysis method are still high. To estimate O_3-associated mortality, we applied CRFs from Turner et al., (2016) and Jerrett et al., (2009), both of which were based on the American Cancer Society Cancer Prevention Study II data. The mortality estimates under the Turner et al., (2016) CRFs were much higher than those from the Jerrett et al., (2009) study due to the elevated hazard ratio (HR) for respiratory mortality and newly observed HR for CVD mortality. A previous study has discussed the advantages of using the new model to estimate O_3-associated respiratory mortality impacts (Malley et al., 2017). Meanwhile, our estimates for all-cause and CVD mortality under the CRFs from the Turner et al., (2016) study could also be informative to policy makers in California, since studies have suggested a stronger positive association between O_3 exposure and ischemic heart disease (IHD) mortality in California than other regions (Jerrett et al., 2013; Turner et al., 2016). Nevertheless, we recognize that knowledge on the CVD mortality due to O_3 exposure is still scarce and inconclusive, and several previous epidemiology and toxicology studies reported attenuated or no associations between O_3 and total mortality (Lipsett et al., 2011; Carey et al., 2013; Atkinson et al., 2016). The U.S. EPA also concluded that the existing evidence is “suggestive of a causal relationship between long-term exposure to O_3 and cardiovascular effects” (U.S. EPA, 2013). Therefore, the total air pollution health burden may be reduced if O_3-associated CVD mortality is excluded. However, such uncertainties in the O_3-associated mortality are less likely to affect our main conclusion that overall, distant emissions lead to greater mortality burdens of air pollution exposure in California than local anthropogenic emissions, as shown in Fig. 3c.

In addition to highlighting the O_3-associated mortality, we also found that GEMM resulted in a greater $PM_{2.5}$-associated mortality burden in California than the more conventional log-linear function and the IER. This could be partly due to the refined exposure models at finer scales, as supported by recent studies (Pinault et al., 2016; Yin et al., 2017; Chen et al., 2017; Eze et al., 2015). Nevertheless, the true relationship between mortality and $PM_{2.5}$ exposure has not been established, although GEMM is intended to relax some assumptions required by previous models. Therefore, the uncertainties and discrepancies among the existing CRFs in the literature are a limitation of the current study and warrant further research. With this in mind, our reported mortality burdens should always be interpreted as potential health benefits from future environmental and public health policies, rather than the "true" death due to bad air quality. Despite the caveats discussed above, our finding indicates that $PM_{2.5}$ originating from California in-state anthropogenic sources is a major contributor to the air pollution-related disease burden in the region and deserves continuous mitigation efforts.

Previous studies have reported the important contribution of distant emissions to air quality and health using global models (Zhang et al., 2017; Anenberg et al., 2014; West et al., 2009). However, few studies have quantified the relative impacts of local vs. nonlocal emissions on human health at a regional scale. Here, we found that in California, whereas the ambient $PM_{2.5}$ concentrations contributed by in-state anthropogenic emissions and by distant emissions from outside of the western United States were comparable, in-state emissions led to much more $PM_{2.5}$-associated deaths. This is because in-state emission-induced $PM_{2.5}$ is mainly concentrated in populous areas such as Los Angeles and San Francisco. The overlapping of dense populations and high pollution concentrations makes in-state emissions the leading cause of $PM_{2.5}$-associated mortality in California. We have reported the similar metropolitan effect of local emissions in a previous study (Wang et al., 2016). Aside from the two metropolitan areas listed above, San Joaquin Valley in central California was also severely affected by in-state emissions (Fig. 4a). In fact, the San Joaquin Valley is among the regions with the worst air quality in the United States, driven by complex interactions between meteorology, emissions, and the terrain (Chow et al., 2006). Tailpipe emissions from heavy-duty diesel trucks along the major freight corridors contributed greatly to the ambient $PM_{2.5}$ in the valley. Ammonia emissions from the agriculture and dairy industry are also responsible for the secondary formation of $PM_{2.5}$ in the valley (Chen et al., 2007; Horowitz et al., 2016).

Being different from $PM_{2.5}$, California ambient O_3 was dominiated by "background O_3" from nonlocal emissions. This finding is consistent with many previous studies (Emery et al., 2012; Fiore et al., 2014; Huang et al., 2015). A recent study concluded that background O_3 contributes 77% of the total O_3 concentrations in California and Nevada in June-July (Huang et al., 2015), which is very close to our estimate (77.2% of background O_3 in July). Among all background O_3 sources, we found that transboundary emissions out of the western United States have the greatest contribution. This is consistent with previous findings that pollution from Asia and Europe via long-range transport contributes substantially to the background O_3 concentration in the western United States (Cooper et al., 2010; Fiore, 2002). The impact of California in-state anthropogenic emissions on ambient O_3 is complex as a result of the photochemical regime that differs with respect to different

regions and seasons (Downey et al., 2015; Jin et al., 2013). The negative mortality estimates shown in Fig. 3 occur mainly because local anthropogenic emissions contributed negatively to O_3 concentrations in winter (Fig. 2b), especially in urban centers such as Los Angeles (Fig. 5b and Supplementary Information Table S6). Due to the NMVOC-limited photochemical regime in winter, our model estimated that wintertime local emissions might reduce ambient O_3. Previous measurement and modeling data (Chinkin et al., 2003; Kim et al., 2016) have reported similar trends. However, since O_3 is a major summertime air pollutant in California, our findings also suggest that future mitigation policies in California should consider the spatial and seasonal O_3 pattern. Local efforts could still be effective by developing additional seasonal-specific policies and regulations targeting O_3-forming pollutants from local emission sources (e.g., vehicles, electricity generation facilities, and refineries) in summer.

The influence of distant emissions to California and other areas of North America has been reported before, but mainly from a purely air pollution perspective. We now find that distant emissions contribute more to air pollution-related mortalities in California than local anthropogenic emissions. However, per unit pollutant, distant sources' impact on mortality was much smaller than that of local sources (i.e., number of deaths per 1% contribution to statewide average $PM_{2.5}$ or O_3). This is mainly because $PM_{2.5}$ and O_3 contributed by the distant emissions group were more evenly distributed across the state, while $PM_{2.5}$ and O_3 contributed by in-state anthropogenic emissions were concentrated in metropolitan areas. Other studies have also suggested that the chemical composition difference would also make $PM_{2.5}$ from distant emissions less toxic than that from local emissions (Hoek et al., 2013; Valavanidis et al., 2008), which could also affect the mortality impacts of different emission groups but is beyond the scope of our study. Overall, the total environmental and public health impacts from distant emissions have long been neglected in California and many other jurisdictions, which suggests that governments and policy makers need to consider international collaborations to manage air quality and public health problems in the long term. For local governments, we confirm that for $PM_{2.5}$, reducing in-state anthropogenic emissions is the most efficient way to meet its national ambient air quality standard. For O_3, we underline the importance of seeking international collaborations. We also want to emphasize the spatial and seasonal variations of ambient air pollutants due to local emissions. Developing more seasonal-specific policies to reduce local emissions related to winter $PM_{2.5}$ and summer O_3 would further improve air quality and protect public health in California.

5. Conclusions

In this study, we investigate the contributions of local and nonlocal emission sources to ambient $PM_{2.5}$ and O_3 and their associated mortality in California using a geographic source apportionment model and existing epidemiological evidence. We report that air pollutants due to distant emissions lead to more deaths in California than local anthropogenic emissions. This is primarily due to the enhanced O_3 concentrations and its mortality burden estimates attributable to distant emissions. These results suggest that substantial health benefits associated with O_3 control could have been underestimated before, and local

governments should work collaboratively with international counterparts to develop emission control policies in the future.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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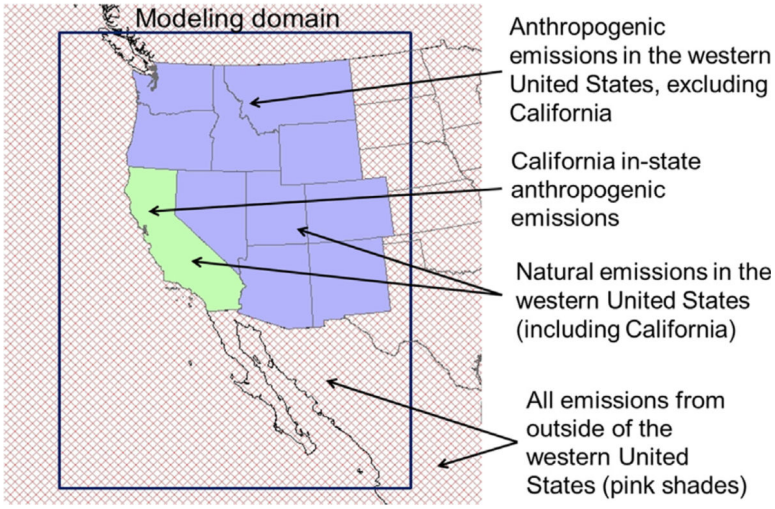

Fig. 1.

Four emission groups that affect California's air quality: (1) California in-state anthropogenic emissions; (2) anthropogenic emissions from California's neighboring states in the western United States, including Washington, Oregon, Nevada, Arizona, Idaho, Utah, Montana, Wyoming, Colorado, and New Mexico; (3) natural emissions in the western United States; and (4) all emissions from outside of the western United States.

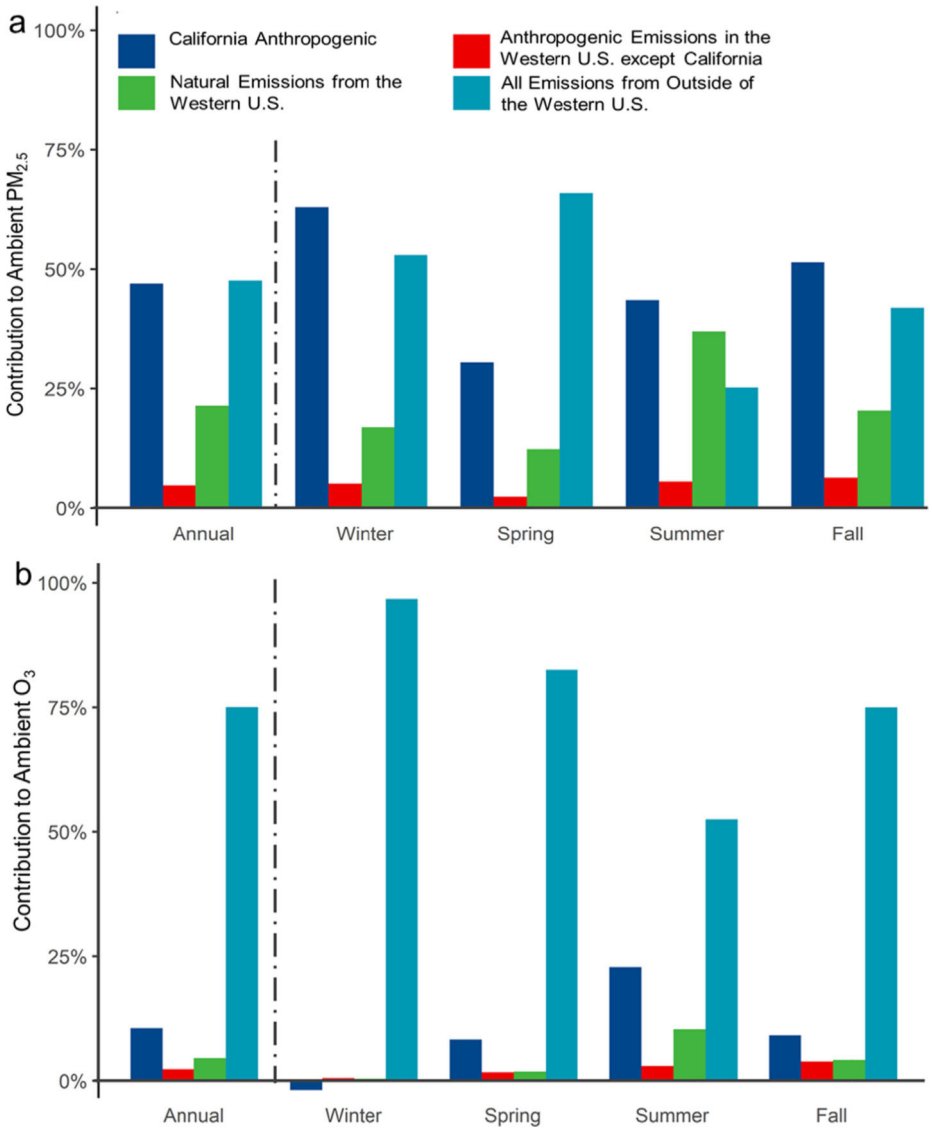

Fig. 2.

Relative contribution of different emission groups to California's ambient (a) $PM_{2.5}$ and (b) O_3 in different seasons in 2012.

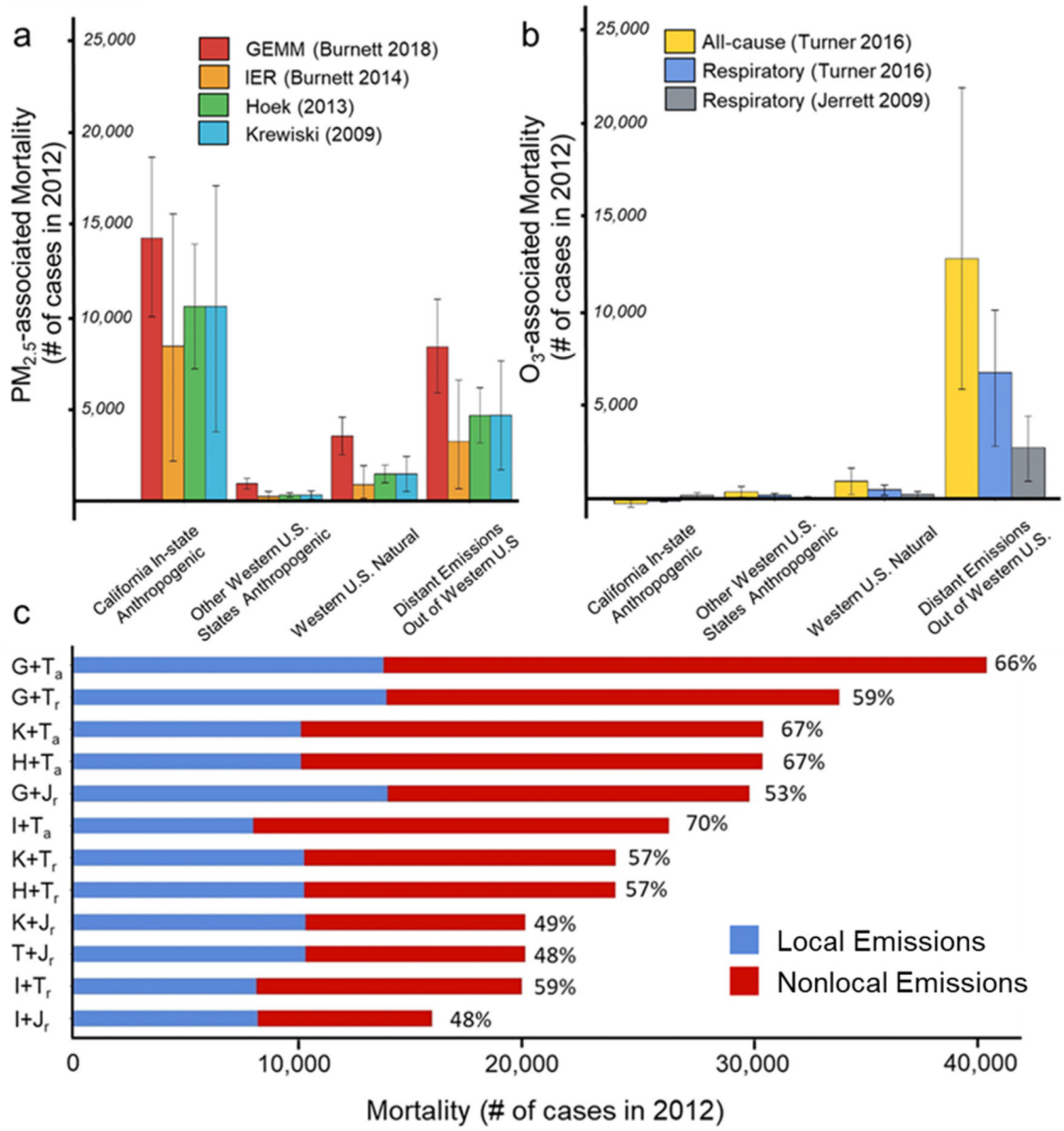

Fig. 3.

(a) $PM_{2.5}$- and (b) O_3-associated mortality burdens in California in 2012 by emission groups under different concentration response functions (CRFs). (c) Total air pollution-related mortality burdens in California with respect to CRF combinations and the relative contribution of nonlocal emissions to total mortality in percentage. On the Y-axis of (c), each combination includes a $PM_{2.5}$ CRF (front) + an O_3 CRF (back). $PM_{2.5}$ CRFs include GEMM (G), Krewski (2009) (K), Hoek (2013) (H), and IER (I); O_3 CRFs include all-cause mortality from Turner (2016) (T_a), respiratory mortality from Turner (2016) (T_r), and respiratory mortality from Jerrett (2009) (J_r). Local Emissions refers to the California In-state anthropogenic group in (a) and (b), and Nonlocal Emissions includes the other three emissions groups.

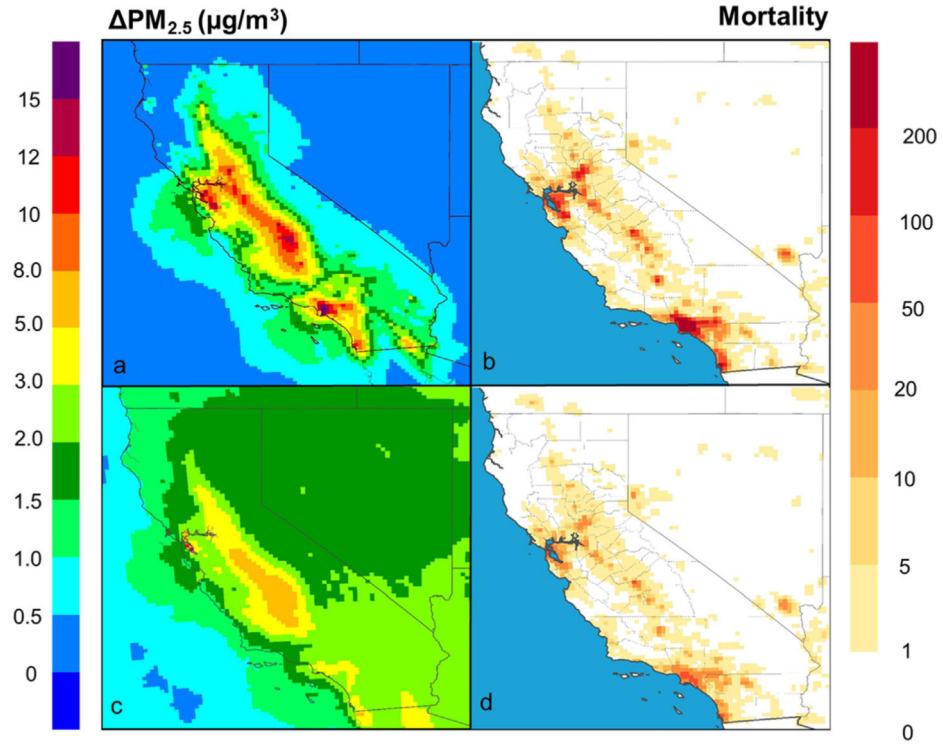

Fig. 4.

Spatial distribution of the contributions of California in-state anthropogenic emissions (a, b) and emissions from outside of the western United States (c, d) to ambient $PM_{2.5}$ concentrations (a, c) and the associated mortality (b, d).

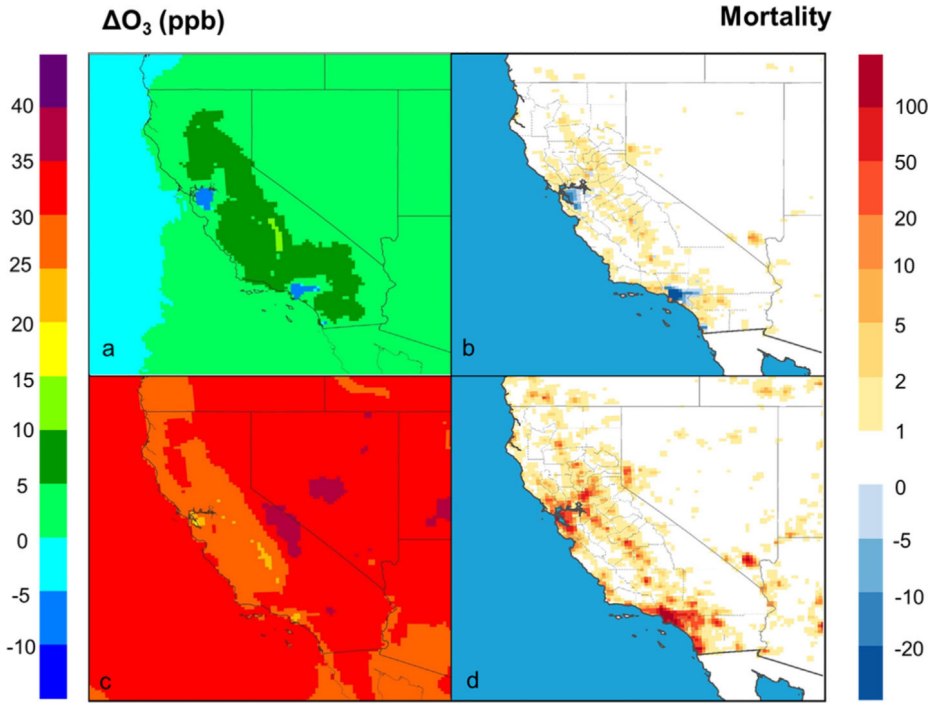

Fig. 5.

Spatial distribution of the contributions of California in-state anthropogenic emissions (a, b) and emissions from outside of the western United States (c, d) to ambient O_3 concentrations (a, c) and the associated mortality (b, d).